CASE REPORT

Acute Pancreatitis, When the Cause is Hypertriglyceridaemia: A Case Report

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Introduction:

Patients with very high levels of serum triglycerides are at risk for pancreatitis. The pathophysiology of the condition is not certain, and some pancreatitis never develops in patients with very high serum triglyceride (TG) levels. Most patients with congenital abnormalities in TG metabolism present in childhood but may not be diagnosed for several years. People with hereditary pancreatitis usually have the typical symptoms that come and go over time. Episodes last from two days to two weeks. A determining factor in the diagnosis of hereditary pancreatitis is two or more family members with pancreatitis in more than one generation. But hypertriglyceridaemia-induced pancreatitis first presenting in adults is more common due to acquired problem in lipid metabolism.

Although there are no clear TG levels that predicts pancreatitis, it is more common when TG level is more than 500 mg/dl. The risk of pancreatitis may be more related to the TG level following consumption of a fatty meal as postprandial increases in TG are inevitable if fat containing foods are eaten. Fasting TG level levels in persons prone to pancreatitis should be kept well below that level. Here hyperlipidaemia and acute pancreatitis is elaborated because such case is not very common but interrelated[1,2,3,5].

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Case Report:

Mrs. AA, a 34 yrs old woman, hailing from Dhaka admitted into Holy Family Red Crescent Medical College Hospital with severe constant upper abdominal pain for two days. Pain aggravated during taking heavy meal and slightly decreased while leaning forward and taking NSAID. She also complained of vomiting for two days. Vomiting was not projectile, not self induced, not blood stained and vomiting did not relieve symptoms. She had no history of taking steroid, alcohol, thiazide diuretic, sodium valproate but history of taking metformin, glicazide, fenofibrate and thyroxine. There was no history of trauma or surgery or same attack previously. She was a known case of hypothyroidism and diabetes mellitus.

On examination, the pt was found anxious looking, liked to be in leaning forward and dehydrated. Crops of yellow papules were found mainly in extensor surface of extremities and over large joints (both knee joint and elbow joint). Her Blood pressure was-110/90 mm of Hg, pulse 84/minute, Temperature 98^0 F, respiratory rate 20 breaths/ minute. On palpation, epigastric tenderness

was present but there was no muscle guard, murphy's sign was negative and liver was just palpable. On auscultation, bowel sound was present but sluggish. Other examination findings revealed no abnormality.

So the provisional diagnosis was acute pancreatitis with hyperlipidaemia. Then some investigations were done. Her Complete Blood Count showed Hb% 14.5g/dl, ESR 30 mm of Hg, total leucocyte count 8.5 k/μL with neutrophil 91.4% and lymphocyte 8.2%. Serum Lipase(2102 U/L) was significantly elevated; Serum Amylase (281U/L) was also raised and Urinary Amylase was 267U/L. USG of whole abdomen was suggestive of a case of acute pancreatitis with peritoneal collection and hepatomegaly with gross fatty change in liver, X-ray abdomen in erect posture was done to exclude perforation of hollow viscus, CT scan of upper abdomen was also suggestive of acute pancreatitis with Peripancreatic fluid collection, Fasting Lipid profile showed highly raised total cholesterol(848 mg/dl) and Triglyceride(5386 mg/dl) level .Random blood sugar was also raised (20.8 mmol/l), Urinary albumin (+) and Sugar (+) was present .Blood Urea (25.3 mg/l) and serum creatinine(0.79mg/dl) was within normal limit. Hypocalcaemia (4.20 mg/dl) was seen and Inorganic phosphate level (1.70mg/dl) was also slightly reduced. Her ECG, Chest X-ray PA view all were found Normal .Serum Electrolytes was within normal limit(Na^+-144.0 mmol/L, Cl^- - 110.1 mmol/L, K^+- 3.7 mmol/L) .Liver function test(Serum bilirubin: 10.60 μmol/L, SGPT: 20.12 IU/L, Alkaline phosphatase: 125.31 IU/L) and Thyroid function test (TSH: 5.58μ IU/ml) showed no abnormality.

After proper diagnosis, treatment was aimed at supportive measures such as withholding food or fluid by mouth (to limit the activity of the pancreas that makes symptoms worse),nasogastric suctioning , fluid replacement by intravenous (IV) infusion, pain relief, broad spectrum antibiotic and Inj. Calcium gluconate (due to decreased calcium level and features of tetany).

Discussion:

Acute pancreatitis accounts for 3% of all cases of abdominal pain admitted to hospital. Acute pancreatitis occurs more often in men than women. Most common causes of acute pancreatitis are gallstones, alcohol, idiopathic and post ERCP. But hypertriglyceridaemia is a rarer cause of acute pancreatits[4, 6, 7].

In hypertriglyceridaemia there is local release of free fatty acid and lysolecithin from lipoprotein substrates in the capillary bed of pancreas. When the concentration of lipids exceeds the binding capacity of albumin, they lyses membranes of parenchymal cells, initiating a chemical pancreatitis.

Considering the evaluation of the present case, for example, pattern of eruption of skin lesion (papules) that develops in extensor surface of extremities and large joints and hepatomegly, this case belong to hyperlipidaemia. In addition he had constant upper abdominal pain which radiated to back suggestive of acute pancreatitis.

The primary therapy for high TG levels is diet restriction, avoidance of alcohol, simple sugars, refined starches, saturated and trans fatty acids, and restricting total calories. Control of secondary causes of high TG levels may also be helpful. In patients with fasting triglycerides more than 500 mg/dl despite adequate dietary compliance and certainly in those with a previous episode of pancreatitis,

therapy with triglyceride lowering drugs (a fibric acid derivative or an HMG-Co A reductase inhibitor) is indicated[2].

The initial management of acute pancreatitis is based upon analgesic using pethidine, and correction of hypovolaemia using normal saline and/or colloids. Nasogastric aspiration is only necessary if paralytic ileus is present Prophylactic broad spectrum intravenous antibiotics such as imipenem or cefuroxime may improve outcome in severe cases. Hypoxic patients need oxygen and patients who develop adult respiratory distress syndrome (ARDS) may require ventilatory support. Hyperglycemia is corrected by insulin, but it is not necessary to correct hypocalcaemia by IV calcium injection unless tetany occurs. All severe cases should be managed in high dependency or intensive care units[8, 9, 10].

With supportive measures, patient's condition gradually improved and he was discharged from hospital after ten days. Then the patient came in outdoor after seven days. At that time check USG and lipid profile was done. USG showed formation of pseudocyst (3 cm in diameter) but lipid profile was within normal limit. Then the patient was advised to come after two weeks .Then USG was done again and was revealed normal study. So this cyst disappeared spontaneously in course of time and patient recovered completely with this conservative treatment.

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